Annals of Clinical and Analytical Medicine
Original Research

Antibiotics resistance and effects on atopic dermatitis of skin bacteria in 7A Military Hospital

Skin bacteria on atopic dermatitis patients

Dinh-Duy Pham[1], Luu Thi Thuy Hai[2], Manh-Kien Nguyen[3], Phu-Suong Nguyen[3], Thanh-Cong Nguyen[4], Hoang-Dung Tran[4], Quang-Tri Le[5], Ngoc- Le Nguyen[3]
[1]Department of Infectious Disease, 7A Military Hospital, Ho Chi Minh City, [2]School of Agriculture and Aquaculture, Tra Vinh University , Tra Vinh Province
[3]Department of Laboratory and Pathology, 7A Military Hospital, Ho Chi Minh City, [4]Faculty of Biotechnology, Nguyen-Tat-Thanh University, Ho Chi Minh City
[5]Department of Orthopedics and Department of High-Tech Diagnostics, 7A Military Hospital, Ho Chi Minh City, Viet Nam

Abstract

Aim: This study aimed to identify the skin bacterial infection in atopic dermatitis patients treated at 7A Military Hospital, and evaluate the infection rate, effect on dermatitis conditions, and antibiotic resistance of S. aureus in the studied patients. The study was expected to serve as references for clinical activities in further researches of atopic dermatitis and related infections.

Materials and Methods: This study employed the in-vitro experimental approach and convenience sampling. The total sample size comprised of 190 isolating samples from 190 patients. The severity of atopic dermatitis was assessed using the SCORAD index. Antibiotic susceptibility test followed the modified Kirby – Bauer method and the 2015 CLSI criteria. Data analysis was performed using the biological statistical algorithm with SPSS 16.0 software.

Results: S. aureus accounted for 76.32% of all samples. The infection rate was higher in longer disease contract, presence of personal or family atopy, and higher recurrence, itchiness, and severity ($p < 0.05$). Resistance was high against clindamycin (77.2%) and erythromycin (76.6%), significant against cefoxitin (49.7%), and low against amikacin (4.1%).

Discussion: The dominance of S. aurerus presence in the isolates and its role as both the cause and consequence of atopic dermatitis was well-known in the medical literature. Considerable cefoxitin resistance signified the presence of methicillin-resistant strain (MRSA), and the resistance against clindamycin and erythromycin was also reported in the literature. Amikacin still maintained its effectiveness and that was corresponding with other medical reports.

S. aureus infection and relationship with atopic dermatitis were explainable by the medical literature. MRSA prevalence and resistance against clindamycin and erythromycin were alarming. Amikacin was still a viable treatment. Infection control, alternative treatments, and drug resistance monitors should be performed rigorously.

Keywords

Staphylococcus Aureus; Atopic dermatitis; Antibiotics resistance; SCORAD; MRSA

DOI: 10.4328/ACAM.20183 Received: 2020-04-20 Accepted: 2020-05-05 Published Online: 2020-05-06 Printed: 2020-06-30 Ann Clin Anal Med 2020;11(Suppl 3): S163-167
Corresponding Author: Quang-Tri Le, Director of 7A Military Hospital, Head of Department of Orthopedics, Head of Department of High-Tech Diagnostics, 466 Nguyen Trai Street, Ward 8, District 5, Ho Chi Minh City, 72706, Viet Nam.
E-mail: lqtri@ntt.edu.vn / tsbstri@yahoo.com P: (+084)0839241868 / (+084)0913126229
Corresponding Author ORCID ID: https://orcid.org/0000-0002-2777-0828

Introduction

Atopic dermatitis (AD) is a common chronic, recurring skin inflammation with typical symptoms of pruritus, skin dryness, and other skin lesions. The pathogenesis is complex and still somewhat unclear, amongst which immunity disorders and cutaneous infections play a significant role. The condition usually develops in early childhood, as 60% of patients have symptoms before one year of age, and about 10 – 20% of the pediatric population suffered from this disease. In the U.S., the disease affects 11.3 – 12.7% of the adults and 6.9 – 7.6% of the children. In the U.K., 10.6% of children aged 13-14 years and 8.1% of the adults have this disease, and in Denmark, the number is 21.3% and 10%, respectively [1,2]. In the Asia-Pacific, the 12-month prevalence of AD in children aged 13-14 years ranges from 0.9% in China to 9% in Malaysia and Singapore [3]. Impaired skin immunity in AD patients is expected to facilitate microbial infection, especially the bacterium Staphylococcus aureus; up to 90% cases of AD had S. aureus infection. The bacterium, in turn, also exacerbates dermatitis and facilitates the virulence and toxicity of bacterial infection in general [4]. To make matters worse, antibiotics resistant strains start emerging especially the infamous methicillin-resistant strains (MRSA) [5]. Identification of skin bacteria on atopic dermatitis and their drug resistance for selection of proper treatment is, consequently, essential. As a result, this study was performed on atopic dermatitis patients in 7A Military Hospital to fulfill the listed below goals:

1. Identify the skin bacteria in the patients.
2. Assess the infection rate of skin S. aureus which was expected to dominate the skin bacteria.
3. Assess S. aureus effect on the illness severity and its antibiotic resistance.

The results are expected to serve as reference data for clinical activities and contribute to the basis for further insights and researches on this issue.

Material and Methods

Date, location, experimental objects, and participants

This study was performed on 190 isolating samples harvested from pathologic skin scales of 190 atopic dermatitis patients treated at 7A Military Hospital from July 2017 to December 2019. The diagnosis of atopic dermatitis was based on Hanifin and Rajka (1980) [6, 7]. Patients aged less than 2 months, patients with systemic diseases, diabetes, mental conditions, and other underlying conditions were excluded from the study. Patient participation was strictly voluntary.

Study design and procedures

This study employed the in-vitro experimental approach and convenience sampling. The total sample size comprised of 190 isolating samples from 190 patients. The severity of atopic dermatitis was assessed using the SCORAD index [8].

The sampling was done at the lesion sites. The area surrounding the lesion was cleaned and sterilized with ethanol. The lesion was cleaned with gauze and saline. The sterilized cotton swabs were used to collect lesion pus, debris, or underlying tissue samples for bacteria isolation. Bacteria isolated from the samples were identified and tested for antibiotic resistance using the modified Kirby – Bauer method [9] and the 2015 CLSI criteria (M100-S25 Performance Standards for Antimicrobial Susceptibility Testing; Twenty-Fifth Informational Supplement). Data analysis was performed using the biological statistical algorithm with SPSS 16.0 software.

Ethical declaration

Participation in this study was strictly voluntary. The participants were informed of the study beforehand. Participant privacy was guaranteed, personal and pathologic data of the participants were kept in secret using computer encoding. The agreement of parents or guardians was strictly required for the participation of patients below 15 years of age. The participants were guaranteed to receive compensation for any injuries caused by the study, to have their rights and interests preserved, and to be protected against any discrimination. The participants had the right to withdraw from the study without having any penalties or punishments.

The Medicine Scientific Research Ethics Committee of the 7A Military Hospital approved this study (Number: 83/QĐ-HĐYĐ-BV7A, date: 20.06.2017).

Results

Bacterial presence on patients' skin samples

Amongst the 190 isolating samples from 190 patients treated in 7A Military Hospital from July 2017 to December 2019, 18 had no bacterial isolates (9.47%), and 172 had bacterial isolates (90.53%). There were 145 S. aureus isolates (76.32%), 20 coalugase-negative Staphylococci (CoNS) isolates (10.53%), 5 Streptococcus spp. isolates (2.63%), and 2 other Gram-negative bacilli isolates (1.05%).

Demographics and clinical data of S. aureus infections in atopic dermatitis patients

Genders and geographical dwellings

Amongst the S. aureus infected patients, 71 (49.00%) were males and 74 were females (51.00%). Seventy-seven cases were the city-dwelling patients (53.1%) and 68 cases (79.0%) were rural patients. There was no significant difference between genders or between urban and rural areas ($p > 0.05$).

Ages and occupation

The highest S. aureus infection rate was observed in patients aged under 18 years (55.2%). Adult patients over 18 years old accounted for 44.8%. The average age of S. aureus infected patients was 26.3±2.4 years. Children under school age and students accounted for the highest percentage of occupations (55.2%), workers and farmers made up 25.5% and patients with other occupations accounted for 19.3%. The differences between ages and between jobs were not significant ($p > 0.05$).

Time of disease contract

There was a sudden increase in the percentage of S. aureus infection in patients who has atopic dermatitis for more than one year. The lowest rate was in patients who had atopic dermatitis for less than 6 months (5.5%), followed > 6 - ≤ 12 months (9.7%), >5 years (24.8%), >2 - ≤ 5 years (26.9%), >1 - ≤ 2 years 33.1%. The difference was significant ($x2 = 8.4$, $p < 0.05$).

Personal and family clinical history

The S. aureus infection rate for asthma, allergy, and allergic rhinitis history, and no history patients was 6.2%, 38.6%, 32.4%, 22.8%, respectively. The S. aureus infection rate for a patient

with family asthma, allergy, and allergic rhinitis history, and no history was 6.2%, 38.6%, 32.4%, 22.8%, respectively. Patients with personal and family asthma, allergy, and allergic rhinitis history had a higher S. aureus infection rate (x2 was 8.9 and 23.4, respectively, $p < 0.05$).

Recurrence and symptoms

The majority of patients with S. aureus infection were recurring patients (98.6%), only 2 patients (1.4%) did not experience recurrences. Most S. aureus infection patients had high itchiness (95.2%), only 4.8% reported low itchiness.

Lesion position and condition stage

The infection rate of S. aureus was 88.4% in flexor sites, significantly higher than other areas ($p < 0.05$). There were 18.6% of patients who had lesions in both flexor and extensor sites and only 26.2% of patients had lesions in extensor sites which were more popular in children than adults. S. aureus infection rate was highest in lower limbs (84.9%), followed by arms (60.4%), cheeks (60.0%), body (33.3%). High infection in the limbs was due to high exposure of these areas to the outer environment. S. aureus infection rate was found in 73.1% of acute dermatitis, 20.7% of semi acute dermatitis, and only in 6.2% of chronic dermatitis.

S. aureus infection and the severity of atopic dermatitis

The SCORAD index of patients positive for S. aureus was significantly higher than of negative patients ($p < 0.01$) (data not presented), implying a relationship between the infection and atopic dermatitis severity.

S. aureus antibiotics resistance

Resistance in general

S. aureus isolated in this study had the highest resistance against clindamycin (77.2%) and erythromycin (76.6%). Resistance against cefoxitin – the specific marker for MRSA strains [10] – achieved the third rank (49.7%). Resistance against levofloxacin, ciprofloxacin, trimethoprim+ sulfamethoxazole, chloramphenicol, and gentamycin was from 29.0% to 46.9%. Lowest resistance was observed against amikacin (4.1%) (Table 1).

Resistance per dermatitis severity

S. aureus antibiotics resistance between different dermatitis severity levels was not significantly different in most cases ($p > 0.05$) except for clindamycin in which resistance of medium severity (71.9%) was significantly higher ($p < 0.05$) (Table 2). There was also no significant difference in resistance per lesion location.

Resistance per condition stages

Antibiotic resistance of S. aureus against cefoxitin, clindamycin, levofloxacin, gentamycin, and erythromycin was different in different stages ($p < 0.05$), the highest resistance was observed in the acute stage. The difference in resistance between stages was not significant in the cases of ciprofloxacin, chloramphenicol, and trimethoprime+sulfamethosazole ($p > 0.05$) (Table 2).

Resistance per itchiness level

Antibiotics resistance of S. aureus against clindamycin in high itchiness (69.7%) was 4.7 times higher than in low itchiness ($p < 0.05$), against chloramphenicol in high itchiness (42.0%) was 7.5 times higher than in low itchiness ($p < 0.01$), against gentamycin in high itchiness (42.9%) was 3.3 times higher than

Table 1. S. aureus antibiotic resistance in general

Antibiotics	Susceptible		Intermediate		Resistant		Total	
	n	%	n	%	n	%	n	%
Amikacin	139	95.9	0	0	6	4.1	145	100
Cefoxitin	73	50.3	0	0	72	49.7	145	100
Clindamycin	29	20.0	4	2.8	112	77.2	145	100
Ciprofloxacin	94	64.8	2	1.4	49	33.8	145	100
Chloramphenicol	76	52.4	5	3.4	64	44.1	145	100
Trimethoprim+ Sulfamethoxazole	85	58.6	6	4.1	54	37.2	145	100
Levofloxacin	103	71.0	0	0	42	29.0	145	100
Gentamycin	73	50.3	4	2.8	68	46.9	145	100
Erythromycin	31	21.4	3	2.1	111	76.6	145	100

Table 2. Antibiotic resistance of S. aureus per dermatitis severity and condition stages

Antibiotics	Stages							
	Acute		Semi acute		Chronic		x^2	p
	n	% R	n	% R	n	% R		
Amikacin	6	4.9	2	3.1	1	5.7	0.7	0.7
Cefoxitin	58	52.5	17	32.4	5	25.2	8.3	0.01
Clindamycin	86	76.9	27	51.7	7	28.5	21.6	0
Ciprofloxacin	39	34.3	12	22.6	5	23.2	2.8	0.2
Chloramphenicol	49	44.2	16	31.1	5	30.3	2.4	0.3
Trimethoprim+ Sulfamethoxazole	38	34.1	18	31.9	6	27.7	0.5	0.7
Levofloxacin	38	34.5	9	14.7	5	24.2	6.5	0.04
Gentamycin	55	49.7	14	25.5	9	35.3	8.3	0.01
Erythromycin	85	78.7	24	45.1	8	39.1	16.4	0
	Dermatitis severity							
	Mild		Medium		Severe			
	n	% R	n	% R	n	% R		
Amikacin	0	0.0	5	4.2	3	8.7	3.2	0.2
Cefoxitin	6	31.7	59	44.6	15	42.5	0.8	0.6
Clindamycin	3	18.2	94	71.9	22	61.5	16.8	0
Ciprofloxacin	1	5.2	38	29.3	14	37.8	4.6	0.08
Chloramphenicol	5	25.8	54	41.2	15	38.9	1.6	0.4
Trimethoprim+ Sulfamethoxazole	5	25.9	46	33.9	14	38.9	0.7	0.6
Levofloxacin	2	7.3	35	26.9	12	32.9	3.8	0.1
Gentamycin	2	12.1	52	41.9	17	47.9	5.6	0.1
Erythromycin	7	35.7	89	67.5	23	64.9	5.5	0.05

Table 3. S. aureus antibiotic resistance per itchiness level

Antibiotics	Itchiness level					
	High		Low		OR (95%CI)	p
	n	% R	n	% R		
Amikacin	8	4.9	0	0	0.95 (0.91-0.99)	0.4
Cefoxitin	69	43.8	9	37.4	1.4 (0.5-3.3)	0.5
Clindamycin	111	69.7	8	32.8	4.6 (1.8-11.4)	0.01
Ciprofloxacin	51	32.4	4	14.6	2.9 (0.9-10.0)	0.09
Chloramphenicol	69	42.0	1	9.2	7.6 (1.8-34.4)	0.002
Trimethoprim+ Sulfamethoxazole	56	35.2	8	27.9	1.5 (0.5-3.8)	0.5
Levofloxacin	45	28.2	5	15.6	2.4 (0.7-8.6)	0.2
Gentamycin	68	42.9	5	19.2	3.4 (1.1-10.3)	0.03
Erythromycin	109	68.7	10	42.9	3.1 (1.3-7.6)	0.01

in low itchiness ($p < 0.05$), against erythromycin in high itchiness (68.7%) was 3,0 times higher than in low itchiness ($p < 0.05$). Difference between itchiness level against amikacin, cefoxitin, ciprofloxacin, levofloxacin, trimethoprim + sulfamethoxazole was not significant ($p > 0.5$) (Table 3).

Discussion

S. aureus infection in atopic dermatitis patients

S. aureus was dominant amongst infecting bacteria, accounted for 76.32% samples, and 90.53% positive isolates. The bacterium, in fact, has long been known for its prevalence in atopic dermatitis as 90% of the cases were positive [4, 11]. Dam and Nguyen (2010) report 76.7% presence of S. aureus [12] and Alenizi (2014) observed 65% infection in lesions, compared with 30% in healthy skin and 13.33% in healthy children [13].

The SCORAD index of patients positive with S. aureus was significantly higher than of negative patients ($p < 0.01$) (Figure 1), implying a relationship between the infection and atopic dermatitis severity. This was similar to the studies of Alenizi (2014) [13]. S. aureus infection has long been known as both a consequence and cause of atopic dermatitis; dermatitis resulted in changes in the skin structure and the immunity systems leads to greater adhesion and colonization of S. aureus. The bacteria in turn excessively stimulate immunity responses, express cytotoxicity, and protect other opportunistic pathogens in the lesion sites, which exacerbate the AD. Medical damage such as scratching, which is common due to itchiness, also contributes to bacterial colonization and then further increases AD severity, which explains higher infection with itchiness level. [4] Such a relationship also explains the positive relationship between personal and family allergic atopy with S. aureus infection, as allergy was observed to have a positive relationship with AD [11]. Contract time and recurrence lead to a higher frequency and longer time of skin damages, hence increase bacterial exposure and contribute to the correlation between them and infection rate.

Antibiotic resistance of S. aureus

S. aureus naturally is sensitive to most known antibiotics. However, its extensive antibiotic resistance developed quickly and antibiotics became prominent selective pressure to its co-evolutionary with humans. Amongst the resistant strains is the notorious methicillin-resistant S. aureus (MRSA) which is known for its multidrug resistance (MDR), high diversity, a large scale of infection worldwide, and a considerable rate of morbidity and mortality [5]. The prevalence of MRSA in this study was significant as the resistance against cefoxitin, the specific marker for MRSA, is high (49.7%). Vicetti Miguel et al. (2019) reported 55.7% MRSA amongst available 37070 isolates in 9 years [14]. Saba et al. (2017) detected 17% putative MRSA isolates harvested in hospital public point contacts with significant MRA capability, included one pandrug resistant (PDR) isolate [15]. In atopic dermatitis patients, Jagadeesan et al. (2014) observed 25.21% MRSA in children aged less than 12 years [16], Jung et al. (2015) detected 12.9% MRSA isolates [17].

The bacteria isolated in this study had significant resistance against clindamycin (77.2%) and erythromycin (76.6%). Resistance against the duo and related antibiotics has been detected for long in various cases worldwide as a response to their frequent use both to treat the MRSA as an alternative of methicillin and the methicillin-sensitive strains (MSSA) [18,19]. Some other studies, however, reported, although still significant, a much lower resistance against the above duo [15, 20], hence a wider sampling and longer monitoring are necessary to determine the extent and scale of resistance to the duo and the related drugs.

On the other hand, amikacin resistance of the isolated S. aureus was low (4.1%). High susceptibility to amikacin and vancomycin in both MSSA and MRSA was observed in many studies [18, 21]. Experiments of Broussou et al. (2018) showed that amikacin – vancomycin combo was effective against both planktonic and biofilm S. aureus and could prevent amikacin resistance [22].

Antibiotics resistance was not different in different dermatitis severity except the case against clindamycin. Clindamycin and erythromycin were also significantly less effective in the acute stage and high itchiness, but that had little meaning within this study as the duo was unfavorable overall.

Remarks and recommendations

The high occurrence of S. aureus infection in atopic dermatitis has long been recorded in literature and it is also well known as both the result of and reason for this disease. A positive relationship between infection and AD severity, recurrence, time, and itchiness can be explained within this framework. Prevention and treatment of S. aureus infection, therefore, is essential for the treatment of atopic dermatitis. It should be noticed that the bacteria, including drug-resistant strains, are common nosocomial pathogens and account for considerable morbidity and mortality amongst inpatients [5], hence control and prevention of hospital-acquired infection should be paid enough attention at.

The significant prevalence of MRSA strains, expressed via cefoxitin resistance (49.7%) was worrying due to their notorious MDR capability (especially against β-Lactams) and poor treatment outcome. Popular alternatives of β-Lactams include clindamycin and erythromycin although their extensive use both against MRSA and MSSA also leads to considerable resistance; in this study, the duo was rendered ineffective hence their administration is inadvisable. However, other studies still reported better outcomes; the effects of the duo, therefore, should be monitored and evaluated in researches at larger scales. Other alternatives comprised amikacin and vancomycin, which were still very effective in many studies and this study. Some approaches besides antibacterial agents comprised the antimalarial artemisinin [23], host antibodies or antimicrobial peptides [24, 25] are being researched and showing promise.

Conclusions

This study showed the dominant occurrence of S. aureus infection in atopic dermatitis cases and the positive relationship between the infection and the length of dermatitis contract, personal or family atopy, recurrence, and severity, which was explainable by the medical literature. The bacteria showed strong resistance to clindamycin and erythromycin and a significant prevalence of MRSA strains, which was a worrying issue. Amikacin resistance was low hence the drug could be a potential solution. Prevention of pathogen infection, exploration of other treatment alternatives, and further researches and

monitoring of the bacterial resistance should have higher attention to mitigate S. aureus effects on atopic dermatitis and the severity of AD in general.

Scientific Responsibility Statement
The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement
All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

Funding: *None*

Conflict of interest
None of the authors received any type of financial support that could be considered potential conflict of interest regarding the manuscript or its submission.

References
1. Kim J, Kim BE, Leung D. Pathophysiology of atopic dermatitis: Clinical implications. Allergy Asthma Proc. 2019; 40(2): 84–92.
2. Kowalska-Olędzka E, Czarnecka M, Baran A. Epidemiology of atopic dermatitis in Europe. J Drug Assess. 2019; 8(1): 126–8.
3. Lee BW, Detzel PR. Treatment of Childhood Atopic Dermatitis and Economic Burden of Illness in Asia Pacific Countries. Ann Nutr Metab. 2015; 66(Suppl. 1): S18-24.
4. Nowicka D, Grywalska E. The Role of Immune Defects and Colonization of Staphylococcus aureus in the Pathogenesis of Atopic Dermatitis. Anal Cell Pathol. (Amst). 2018; 2018: 1956403.
5. Chatterjee A, Rai S, Guddattu V, Mukhopadhyay C, Saravu K. Is methicillin-resistant Staphylococcus Aureus infection associated with higher mortality and morbidity in hospitalized patients? A cohort study of 551 patients from South Western India. Risk Manag Healthc Policy. 2018; 11: 243–50.
6. Hanifin JM. Diagnostic criteria for atopic dermatitis: consider the context. Arch. Dermatol. 1999; 135(12): 1551.
7. Hanifin, JM, Rajka G. Diagnostic features of atopic dermatitis. Acta Derm Venereol Suppl. (Stockh). 1980; 59: 44-47.
8. European Task Force on AD. Severity scoring of AD: The SCORAD Index (consensus report of the European Task Force on AD). Dermatology. 1993; 186(1): 23–31.
9. Boyle VJ, Fancher ME, Ross RW Jr. Rapid, modified Kirby-Bauer susceptibility test with single, high-concentration antimicrobial disks. Antimicrob Agents Chemother. 1973; 3(3): 418–24.
10. Fernandes CJ, Fernandes LA, Collignon P, Australian Group on Antimicrobial Resistance. Cefoxitin resistance as a surrogate marker for the detection of methicillin-resistant Staphylococcus aureus. J Antimicrob Chemother. 2005; 55(4): 506-10.
11. Kapur S, Watson W, Carr S. Atopic dermatitis. Allergy Asthma Clin Immunol. 2018; 14(Suppl. 2): 52.
12. Dam TH, Nguyen D.H. Identification of Staphylococcus aureus in skin lesions of children atopic eczema. Vietnam Dermatol J. 2010; 1: 22-26. (in Vietnamese)
13. Alenizi DA. Prevalence of Staphylococcus aureus and antibiotic resistance in children with atopic dermatitis in Arar, Saudi Arabia. J Dermatol Dermatol Surg. 2014; 18(1-2): 22-26.
14. Vicetti Miguel CP, Mejias A, Leber A, Sanchez PJ. A decade of antimicrobial resistance in Staphylococcus aureus: A single center experience. PloS. 2019; 14(2):e0212029.
15. Saba C, Amenyona JK, Kpordze SW. Prevalence and pattern of antibiotic resistance of Staphylococcus aureus isolated from door handles and other points of contact in public hospitals in Ghana. Antimicrob Resist Infect Control. 2017; 6: 44.
16. Jagadeesan S, Kurien G, Divakaran MV, Sadanandan SM, Sobhanakumari K, Sarin A. Methicillin-resistant Staphylococcus aureus colonization and disease severity in atopic dermatitis: a cross-sectional study from South India. Indian J Dermatol Venereol Leprol. 2014; 80(3): 229-34.
17. Jung MY, Chung JY, Lee HY, Park J, Lee DY, Yang JM. Antibiotic Susceptibility of Staphylococcus aureus in Atopic Dermatitis: Current Prevalence of Methicillin-Resistant Staphylococcus aureus in Korea and Treatment Strategies. Ann Dermatol. 2015; 27(4): 398–403.
18. Mama M, Aklilu A, Misgna K, Tadesse M, Alemayehu E. Methicillin- and Inducible Clindamycin-Resistant Staphylococcus aureus among Patients with Wound Infection Attending Arba Minch Hospital, South Ethiopia. Int J Microbiol. 2019; 2019: 2965490.
19. Piątkowska E, Piątkowski J, Przondo-Mordarska A. The strongest resistance of Staphylococcus aureus to erythromycin is caused by decreasing uptake of the antibiotic into the cells. Cell Mol Biol Lett. 2012; 17(4): 633–45.
20. Rağbetli C, Parlak M, Bayram Y, Guducuoglu H, Ceylan N. Evaluation of Antimicrobial Resistance in Staphylococcus aureus Isolates by Years. Interdiscip Perspect Infect Dis. 2016; 2016: 9171395.
21. Vamsi Muni KP, Sreenivasulu RV, Praveen KV, Suresh P. Antibiotic Susceptibility Pattern of Staphylococcus aureus and Methicillin-Resistant Staphylococcus aureus Isolated from Various Clinical Specimens in a Tertiary Care Teaching Hospital, Pondicherry. Indian J Public Health Res. 2019; 10(2): 208-13.
22. Broussou DC, Lacroix MZ, Toutain PL, Woehrlé F, El Garch F, Bousquet-Melou A et al. Differential Activity of the Combination of Vancomycin and Amikacin on Planktonic vs. Biofilm-Growing Staphylococcus aureus Bacteria in a Hollow Fiber Infection Model. Front Microbiol. 2018; 9: 572.
23. Lin L, Mao X, Sun Y, Cui H. Antibacterial mechanism of artemisinin / beta-cyclodextrins against methicillin-resistant Staphylococcus aureus (MRSA). Microb Pathog. 2018; 118: 66–73.
24. Gunasekaran P, Fan M, Kim EY, Shin JH, Lee JE, Son EJ, et al. Amphiphilic Triazine Polymer Derivatives as Antibacterial And Anti-atopic Agents in Mice Model. Sci Rep. 2019; 9(1): 15161.
25. Speziale P, Rindi S, Pietrocola G. Antibody-Based Agents in the Management of Antibiotic-Resistant Staphylococcus aureus Diseases. Microorganisms. 2018; 6(1): 25.

How to cite this article:
Dinh-Duy Pham, Luu Thi Thuy Hai, Manh-Kien Nguyen, Phu-Suong Nguyen, Thanh-Cong Nguyen, Hoang-Dung Tran, Quang-Tri Le, Ngoc- Le Nguyen. Antibiotics resistance and effects on atopic dermatitis of skin bacteria in 7A Military Hospital. Ann Clin Anal Med 2020;11(Suppl 3): S163-167